10.0 REFERENCES

AEHA (U.S. Army Environmental Hygiene Agency), 1986. *Evaluation of S-TEAD Solid Waste Management Units;* AEHA Groundwater Consultation Report No. 38-26-1364-86.

Ammerman, C.B., K.R. Fick, S.L. Hansard II, and S.M. Miller, 1973. *Toxicity of Certain Minerals to Domestic Animals: A Review*; National Feed Ingredients Association, Des Moines, IA.

Atef, M., M.A.M Abo-Norage, M.S.M. Hanafy, and A.E. Agag. 1991. *Pharmacotoxicological Aspects of Nitrate and Nitrite in Domestic Fowls*. British Poultry Science 32:399-404.

Aulerich, R.J., R.K. Ringer, and S. Iwamoto, 1974. *Effects of Dietary Mercury on Mink*; Archives of Environmental Contamination and Toxicology, 2:43-51.

Aulerich, R.J., R.K. Ringer, M.R. Bleavins, and A. Napolitano, 1982. *Effects of Supplemental Dietary Copper on Growth, Reproductive Performance and Kit Survival of Standard Dark Mink and the Acute Toxicity of Copper to Mink*; Journal of Animal Science, 55:337-343.

Azar, A., H.J. Trochimowicz, and M.E. Maxfield, 1973. *Review of Lead Studies in Animals Carried Out at Haskell Laboratory - Two-Year Feeding Study and Response to Hemorrhage Study;* In: *Environmental Health Aspects of Lead: Proceedings, International Symposium*, D. Barth, et al., eds. Commission of European Communities. pp. 199-210.

Baedecker, M.J., D.I. Siegel, P. Bennett, and I.M. Cozzarolli, 1989. *The Fate and Effects of Crude Oil in a Shallow Aquifer, I. The Distribution of Chemical Species and Geochemical Facies*; U.S. Geological Survey Water-Resources Investigation Report 88-4220.

Baes, C.F., III, R.D. Sharp, A.L. Sjoreen, and R.W. Shor, 1984. *A Review and Analysis of Parameters for Assessing Transport of Environmentally Released Radionuclides through Agriculture;* Oak Ridge National Laboratory, Oak Ridge, Tennessee.

Bodek, I., W.J. Lyman, W.F. Reehl, and D.H. Rosenblatt, 1988. *Environmental Inorganic Chemistry*; Pergamon Press, Elmsford, NY (Chapters 4 and 7).

Brown, S.L., and J.E. Rossi, 1989. *A Sample Method for Estimating Dermal Absorption of Chemicals in Water;* Chemosphere, 19(12): 1989-2001.

Bruning-Fann C.S., and J.B. Kaneene. 1993. *The Effects of Nitrate, Nitrite, and N-Nitroso Compounds on Animal Health.* Veterinary and Human Toxicology 35:237-253.

Burt, W.H., 1980. *A Field Guide to the Mammals North America North of Mexico;* Houghton Mifflin, Boston, MA.

California EPA, 1994. *Preliminary Endangerment Assessment Guidance Manual*; Department of Toxic Substances Control, Sacramento, California.

Camardese, M.B., D.J. Hoffman, L.J. LeCaptain, and G.W. Pendleton, 1990. *Effects of Arsenic on Growth and Physiology in Mallard Ducklings*; Environmental Toxicology and Chemistry, 9:785-795.

CEPA, 1993a. *Dichloromethane*; Priority Substances List Assessment Report Health Canada, Canada Communications Group Publishing, Ottawa, Canada.

CEPA, 1993b. *Fluoride*; Priority Substances List Assessment Report Health Canada, Canada Communications Group Publishing, Ottawa, Canada.

CEPA, 1993c. *Xylenes*; Priority Substances List Assessment Report Health Canada, Canada Communications Group Publishing, Ottawa, Canada.

CEPA, 1994. *Chromium*; Priority Substances List Assessment Report Health Canada, Canada Communications Group Publishing, Ottawa, Canada

Costanza, R., L. Wainser, E. Folke, and K. Maler, 1993. *Modeling Complex Ecological Economic Systems;* Bioscience, 43(8).

Cowherd, C., G.E. Muleske, P.J. Englehart, and D.A. Gillette, 1985. *Rapid Assessment of Exposure to Particulate Emissions from Surface Contamination Sites;* EPA/600/8-85/002, U.S. Environmental Protection Agency, Washington, D.C.

Doherty, P.C., R.M. Barlow, and K.W. Angus, 1969. *Spongy Changes in the Brains of Sheep Poisoned by Excess Dietary Copper*; Research in Veterinary Science, 10:303-304.

Donohue (Donohue and Associates and Ebasco Services Incorporated), 1990. *Tooele Army Depot-South Area Known Releases, RCRA Facility Investigation-Phase II, Final Draft Data Collection Quality Assurance Plan;* Prepared for U.S. Army Toxic and Hazardous Materials Agency, Aberdeen Proving Ground, Maryland.

Domingo, J.L., 1994. *Metal-Induced Developmental Toxicity in Mammals: A Review*; Journal of Toxicology and Environmental Health, 42:123-141.

Dragun, J., 1988. *The Soil Chemistry of Hazardous Materials;* Hazardous Materials Control Research Institute, Silver Spring, Maryland.

EA (EA Engineering, Science, and Technology, Inc.), 1988. *Tooele Army Depot Preliminary Assessment/Site Investigation, Final Report, Volume II-South Area;* Prepared for U.S. Army Toxic and Hazardous Materials Agency, Installation Restoration Division, Aberdeen Proving Ground, Maryland.

Ebasco, 1991. *Tooele Army Depot-South Area, Suspected Releases, RCRA Facility Investigation-Phase I, Draft Report;* Prepared for the U.S. Army Toxic and Hazardous Materials Agency, Aberdeen Proving Ground, Maryland.

Environmental Photographic Interpretation Center (EPIC), 1982. *Installation Assessment Tooele Army Depot (South Area), Utah.* EPA Report Number TS-PIC-0066.

ERTEC, 1982. *Assessment of Environmental Contamination, Exploratory Stage, Tooele Army Depot, Tooele, Utah;* Prepared for Tooele Army Depot and U.S. Army Toxic and Hazardous Materials Agency, Aberdeen Proving Ground, Maryland.

Everett, D.S., et al., 1986. *HMX: 13 Week Toxicity Study in Rats by Dietary Administration*; Government Reports & Index No. 26, (NTIS/AD-A171601/8), 1985/1986.

Everitt, B.L., and B.N. Kaliser, 1980. *Geology for Assessment of Seismic Risk in the Tooele and Rush Valleys, Tooele County, Utah;* State of Utah, Department of Natural Resources, Special Studies No. 51.

Fimreite, N. and L. Karstad, 1971. *Effects of Dietary Methyl Mercury on Red-Tailed Hawks;* Journal of Wildlife Management, 35:293-300.

Finley, M. and R.C. Stendall, 1978. *Survival and Reproductive Success of Black Ducks Fed Methyl Mercury*; Environmental Pollution, 16:51-63.

Fitzhugh, O.G., A.A. Nelson, E.P. Laug, and F.M. Kunze, 1950. *Chronic Oral Toxicities of Mercuri-phenyl and Mercuric Salts;* Industrial Hygiene and Occupational Medicine, 2:433-442. In: EPA, 1995. *Great Lakes Water Quality Initiative Criteria Documents for the Protection of Wildlife: DDT, Mercury, 2,3,7,8 TCDD, and PCBs.* (EPA-820-B-95-008), March 1995.

Franson, J.C., L. Sileo, O.H. Pattee, and J.F. Moore, 1983. *Effects of Chronic Dietary Lead in American Kestrels (Falco sparverius)*; Journal of Wildlife Diseases, 19:110-113.

Friberg, L., et al., 1979. *Handbook on the Toxicology of Metals*; Elsevier/North Holland Biomedical Press.

Fries, G.H., 1987. *Assessment of Potential Residues in Foods Derived from Animals Exposed to TCDD-Contaminated Soil;* Chemosphere, 16: 2123-2128.

Gasaway, W.C., and I.O. Buss, 1972. *Zinc Toxicity in the Mallard Duck*; Journal of Wildlife Management, 36:1107-1117.

Gerrard, J.M., and G.R. Bortolotti, 1988. *The Bald Eagle: Haunts and Habits of a Wilderness Monarch;* Smithsonian Institution Press, Washington, D.C.

Giacomelli-Maltoni, G., 1972. *Deposition Efficiency of Monodisperse Particles in Human Respiratory Tract;* AIHA J., 33(9): 603-610.

Gilluly, J., 1932. *Geology and Ore Deposits of the Stockton and Fairfield Quadrangles, Utah;* U.S. Geological Survey Professional Paper 173.

Halliburton NUS, 1992. *Analytical Laboratory Guidebook for Environmental Professionals;* Halliburton NUS Environmental Corporation, Pittsburgh, PA.

Harvey, R.B., L.F. Kubena, T.D. Phillips, and N.D. Heidelbaugh, 1986a. *Validation of Impaired Renal Function Chick Model with Uranyl Nitrate.* Bulletin of Environmental Contamination and Toxicology 36:67-72.

Harvey, R.B., L.F. Kubena, S.L. Lovering, H.H. Mollenhauer, and T.D. Phillips, 1986b. *Acute Toxicity of Uranyl Nitrate to Growing Chicks: A Patholphysiologic Study.* Bulletin of Environmental Contamination and Toxicology 37:907-915.

Heinz, G., 1976. *Methylmercury: Second-Year Feeding Effects on Mallard Reproduction and Duckling Behavior;* Journal of Wildlife Management, 40:82-90.

Hem, J.D., 1970. *Study and Interpretation of the Chemical Characteristics of Natural Water, 2nd ed.*; U.S. Geological Survey Water Supply Paper 1473.

Hoffman, D.J., C. Franson, O.H. Pattee, C.M. Bunck, and H.C. Murray, 1985a. *Biochemical and Hematological Effects of Lead Ingestion in Nestling American Kestrels (Falco sparverius)*; Comparative Biochemistry and Physiology, 80C:431-439.

Hoffman, D.J. et al., 1985b. *Survival, Growth and Accumulation of Ingested Lead in Nestling American Kestrels (Falco sparverius)*; Archives of Environmental Contamination & Toxicology, 14:89-94.

Hood, S.W., D. Price, and K.M. Waddell, 1969. *Hydrologic Reconnaissance of Rush Valley, Tooele County, Utah;* State of Utah, Department of Natural Resources, Technical Publication No. 23.

Howard, P.H. (ed.), 1990. *Handbook of Environmental Fate and Exposure Data for Organic Chemicals, Volumes I, II, III, and IV;* Lewis Publishers, Chelsea, Michigan.

Howard, P.H., et al., 1991. *Handbook of Environmental Degradation Rates*; Lewis Publishers, Inc., Chelsea, MI.

Irwin, L.L., R.J. Guenzal, and T.J. Ryder, 1984. *Pronghorn-Habitat Relationships in the Red Rim Area, South-Central Wyoming, Final Report;* Department of Zoology and Physiology, University of Wyoming, Laramie Wyoming. (unpublished report)

James, L.F., V.A. Lazar, and W. Binns, 1966. *Effects of Sublethal Doses of Certain Minerals on Pregnant Ewes and Fetal Development*; American Journal of Veterinary Research, 27:132.

Johnson, D., Jr., A.L. Mehring, Jr., F.X. Savino, and H.W. Titus, 1962. *The Tolerance of Growing Chickens for Dietary Zinc.* In:Gasaway, W.C., and I.O. Buss. 1972. Zinc Toxicity in the Mallard Duck. Journal of Wildlife Management 36:1107-1117.

Korte, N., 1991. *Naturally Occurring Arsenic in Groundwaters of the Midwestern United States;* Environmental Geology and Water Science, 18(2): 137-141.

Kostecki, 1989. *Petroleum Contaminated Soil, Volume I*; Lewis Publishers, Inc., Chelsea, Michigan.

Kupsh, C.C., R.J. Julian, V.E.O. Valli, and G.A. Robinson, 1991. *Renal Damage Induced by Uranyl Nitrate and Oestradiol-17β in Japanese Quail and Wistar Rats.* Avian Pathology 20:25-34.

Lewis, R.J., 1992. *Sax's Dangerous Properties of Industrial Materials, 8th Edition*; Van Nostrand Reinhold, New York, NY.

Lewis, P.K. Jr., W.G. Hoekstra, and R.H Grummer, 1957. *Restricted Calcium Feeding Versus Zinc Supplementation for the Control of Parakeratosis in Swine*; Journal of Animal Science, 16:578-589.

Mehring, A.L. Jr., J.H. Brumbaugh, A.J. Sutherland, and H.W. Titus, 1960. *The Tolerance of Growing Chickens for Dietary Copper;* Poultry Science, 39:713-719.

Mitchell, G., 1990. Department of Veterinary Science, University of Wisconsin, Madison, Wisconsin (personal communication).

Mitsumori, K., K. Maita, T. Saito, S. Tsuda, and Y. Shirasu, 1981. *Carcinogenicity of Methylmercury Chloride in ICR Mice: Preliminary Note on Renal Carcinogenesis*; Cancer Letters, 12:305-310.

Montgomery, J.H., and L.J. Welkom, 1989. *Groundwater Chemicals Desk Reference;* Lewis Publishers, Chelsea, Michigan.

Montgomery, J.H., 1991. *Groundwater Chemicals Field Guide;* Lewis Publishers, Chelsea, Michigan.

Montgomery, James M., Consulting Engineers, 1987. *U.S. Army Corps of Engineers Final Groundwater Quality Assessment Engineering Report, Tooele Army Depot, Utah;* Prepared for the U.S. Army Toxic and Hazardous Materials Agency, Aberdeen Proving Ground, Utah.

Mullins, J.A., R.F. Carsel, J.E. Scarbrough, and A.M. Ivery, 1993. *PRZM-2, A Model for Predicting Pesticide Fate in the Crop Root and Unsaturated Soil Zones: Users Manual for Release 2.0;* EPA/600/R-93/046, U.S. Environmental Protection Agency, Office of Research and Development, Washington, D.C.

National Academy of Sciences (NAS), 1974a. *Chromium*; Committee on Biologic Effects of Atmospheric Pollutants, Division of Medical Sciences, National Research Council, Washington, D.C.

National Academy of Science (NAS), 1974b. *Nutrient Requirements of Domestic Animals. Nutritional Requirements of Dogs*; Subcommittee on Dog Nutrition, National Research Council, Washington D.C.

National Library of Medicine. *Toxicology Data Network Hazardous Substances Data Bank* (Database); NLM, Washington, D.C. (accessed March, 1994)

Ney, R.E. 1990. *Where Did That Chemical Go?- A Practical Guide to Chemical Fate and Transport in the Environment;* Van Nostrand Reinhold, New York.

Nicholson, J.K. and D. Osborn, 1984. *Kidney Lesions in Juvenile Starlings Sturnus Vulgaris Fed on a Mercury-Contaminated Synthetic Diet*; Environmental Pollution (Series A), 33:195-206.

Norton, S.B., and others, 1992. *A Framework for Ecological Assessment at the EPA;* Environmental Toxicology and Chemistry, 2: 1663-1672.

NRC (U.S. Nuclear Regulatory Commission), 1992. *Residual Radioactive Contamination from Decommissioning, Technical Basis for Translating Contamination Levels to Annual Total Effective Dose Equivalent, Final Report;* NUREG/CR-5512, PNL-1994, Vol. 1, U.S. Nuclear Regulatory Commission, Washington, D.C.

NUS Corporation, 1987. *Draft Interim Facility Assessment, Tooele Army Depot-South Area;* Prepared for U.S. Environmental Protection Agency, Contract No. 68-01-7310.

Opresko, D.M., B.E. Sample, and G.W. Suter II, 1993. *Toxicological Benchmarks for Wildlife*; Environmental Restoration Program, Oak Ridge National Laboratory, TN, (ES/ER/TM-86).

ORNL (Oak Ridge National Laboratory), 1992. *Assessment of Location-Specific Applicable or Relevant and Appropriate Requirements (ARARs) for Tooele Army Depot, North and South Areas;* Prepared for U.S. Army Toxic and Hazardous Materials Agency, Aberdeen Proving Ground, Maryland.

Pao, E.M., 1982. *Foods Commonly Eaten by Individuals: Amount Per Day and Per Eating Occasion;* U.S. Department of Agriculture, Human Nutrition Information Service, Consumer Nutrition Center, Hyallsvill, Maryland. Home Economics Research Report No. 44.

Paternain, J.L., J.L. Domingo, A.R. Ortega, and J.M. Llobet, 1989. *The Effects of Uranium on Reproduction, Gestation, and Postnatal Survival in Mice.* Ecotoxicology and Environmental Safety 17:291-296.

Patnaik, P., 1992. *A Comprehensive Guide to the Hazardous Properties of Chemical Substances;* Van Nostrand Publishers, New York.

Pattee, O.H., 1984. *Eggshell Thickness and Reproduction in American Kestrels Exposed to Chronic Dietary Lead*; Archives of Environmental Contamination and Toxicology, 13:29-34.

Pendleton, B.A., B.A. Millsap, K.W. Cline, and D.M. Bird, 1987. *Raptor Management Technique Manual;* Scientific and Technical Series No. 10, National Wildlife Federation, Washington D.C.

Peterson, R.T., 1987. *Field Guide to North American Birds;* Houghton Mifflin Co., Boston, MA.

Robinson, G.A., D.C. Wasnidge, and F. Floto, 1984. *A Comparison of the Distributions of the Actinides Uranium and Thorium with the Lanthanide Gadolinium in the Tissues and Eggs of Japanese Quail: Concentrations of Uranium in Feeds and Foods.* Poultry Science 63:883-891.

Roberson, R.H. and P.J. Schaible, 1960. *The Tolerance of Growing Chicks for High Levels of Different Forms of Zinc.* Poultry Science 39:893-896. In: Gasaway, W.C., and I.O. Buss. 1972. Zinc Toxicity in the Mallard Duck. Journal of Wildlife Management 36:1107-1117.

RTECs, 1996. *Registry of Toxic Effects of Chemical Substances, March 1996;* Citing: Archives of Environmental Health, 23:102. 1971.

RTECs, 1997. *Registry of Toxic Effects of Chemical Substances, March 1997;* Citing: Journal of Toxicology and Environmental Health, 15:609. 1985.

RTECs, 1997. *Registry of Toxic Effects of Chemical Substances, March 1997;* Citing: Pharmacolgical Chemistry Journal, 14:162. 1980.

RTECs, 1997. *Registry of Toxic Effects of Chemical Substances, March 1997;* Citing: Arch.Exp. Pathol. Pharmakol, 18:218. 1984.

RTECs, 1997. *Registry of Toxic Effects of Chemical Substances, March 1997;* Citing: Food Agiculture Organ UN Rep Series, 48:A86. 1970.

RTECs, 1997. *Registry of Toxic Effects of Chemical Substances, March 1997;* Citing: NTIS PB91-185975.

RTECs, 1997. *Registry of Toxic Effects of Chemical Substances, March 1997;* Citing: National Toxicological Progress Technical Report Series NIH-91-3122.

RTECs, 1997. *Registry of Toxic Effects of Chemical Substances, March 1997;* Citing: Prehled.Prumyslove.Toxikol.Org.Latky, 1986:86. 1986.

RTECs, 1997. *Registry of Toxic Effects of Chemical Substances, March 1997;* Citing: Journal of Environmental Science and Health, 17:205. 1982.

RTECs, 1997. *Registry of Toxic Effects of Chemical Substances, March 1997;* Citing: Arch.Toxicology Supplement, 2:371. 1979.

RTECs, 1997. *Registry of Toxic Effects of Chemical Substances, March 1997;* Citing: Q.J. Pharm. Pharmacol, 7:205. 1934.

RTECs, 1997. *Registry of Toxic Effects of Chemical Substances, March 1997;* Citing: Arch. Exp. Pathol. Pharmacol, 97:86. 1923.

RTECs, 1997. *Registry of Toxic Effects of Chemical Substances, March 1997;* Citing: Toxical. Applied Pharmacology, 29:348. 1974.

RTECs, 1997. *Registry of Toxic Effects of Chemical Substances, March 1997;* Citing: Journal of Environmental Science and Health Part B, 18:333. 1983.

RTECs, 1997. *Registry of Toxic Effects of Chemical Substances, March 1997;* Citing: Neurobehaviorial Toxicology, 1:199. 1979.

RTECs, 1997. *Registry of Toxic Effects of Chemical Substances, March 1997;* Citing: Environmental Health Perspective, 46:127. 1982.

RTECs, 1997. *Registry of Toxic Effects of Chemical Substances, March 1997;* Citing: Gg.Sanit, 48:10. 1983.

RTECs, 1997. *Registry of Toxic Effects of Chemical Substances, March 1997;* Citing: Archives of Environmental Health, 23:102. 1971.

RTECs, 1997. *Registry of Toxic Effects of Chemical Substances, March 1997;* Citing: Neurotoxicology, 2:567. 1981.

RTECs, 1997. *Registry of Toxic Effects of Chemical Substances, March 1997;* Citing: Pediatric Research, 36:811. 1994.

RTECs, 1997. *Registry of Toxic Effects of Chemical Substances, March 1997;* Citing: Teratology, 19:41A. 1979.

RTECs, 1997. *Registry of Toxic Effects of Chemical Substances, March 1997;* Citing: Neurotoxicology. Teratol, 10:525. 1989.

RTECs, 1997. *Registry of Toxic Effects of Chemical Substances, March 1997;* Citing: Toxicologist, 5:62. 1985.

RTECs, 1997. *Registry of Toxic Effects of Chemical Substances, March 1997;* Citing: National Toxicological Program Tech Rep. Series NTP-TR-371. 1990.

RTECs, 1997. *Registry of Toxic Effects of Chemical Substances, March 1997;* Citing: Environmental Research, vol 49:93. 1989.

RTECs, 1997. *Registry of Toxic Effects of Chemical Substances, March 1997;* Citing: Drug Chemistry Toxicology, 17:317. 1994.

Rust E&I, 1992. *Tooele Army Depot-South Area, Final Draft Phase II RCRA Facility Investigation Report, Known Releases SWMUs 13 and 17, Volumes I, II, and III;* Prepared for U.S. Army Toxic and Hazardous Materials Agency, Aberdeen Proving Ground, Maryland.

Rust E&I, 1993. *Tooele Army Depot-North Area, Final Preliminary Baseline Risk Assessment*; Prepared for U.S. Army Environmental Center, Aberdeen Proving Ground, Maryland.

Rust E&I, 1994. *Tooele Army Depot North Area Final Remedial Investigation Report for Operable Units 4-10;* Prepared for U.S. Army Environmental Center, Aberdeen Proving Ground, Maryland.

Rust E&I, 1996. *Draft Site-Wide Ecological Risk Assessment for Tooele Army Depot-North Area*; Prepared for the U.S. Army Environmental Center, Aberdeen Proving Ground, MD.

Salhotra, A.M., P. Mineart, S. Sharp-Hansen, and T. Allison, 1990. *Multimedia Exposure Assessment Model (MULTIMED) for Evaluating the Land Disposal of Wastes-Model Theory;* U.S. Environmental Protection Agency, Environmental Research Laboratory, Athens, Georgia.

Sax, N.I. and R.J. Lewis, 1987. *Hawley's Condensed Chemical Dictionary, 11th ed.;* Van Nostrand Reinhold Publishers, New York.

Scanlon, B.R., 1994. *Water and Heat Fluxes in Desert Soils;* Water Resources Research, 30(3): 709-719 and 721-733.

SCS (U.S. Soil Conservation Service), 1992. *Soil Survey of Tooele County Area, Utah;* U.S. Department of Agriculture, Soil Conservation Service, Salt Lake City, Utah. (unpublished data)

Schlicker, S.A. and D.H. Cox, 1968, *Maternal Dietary Zinc, and Development and Zinc, Iron, and Copper Content of the Rat Fetus;* Journal of Nutrition, 95:287-294.

Schroeder H.A., M. Kanisawa, D.V. Frost, and M. Mitchener, 1968. *Germanium, Tin and Arsenic in Rats. Effects on Growth, Survival, Pathological Lesions and Life Span*; Journal of Nutrition, 96:37.

Smith, L.H., W.M. Haschek and H. Witschi, 1980. *Acute Toxicity of Selected Crude and Refined Shale Oil- and Petroleum-derived Substances;* In: W.H. Griest, M.R. Guerin and D.L. Coffin, eds., *Health Effects Investigation of Oil Shale Development*; Ann Arbor Press, Ann Arbor, MI., pp 141-160. In: Stubblefield, W.A., G.A. Hancock, W.H. Ford, and R.K. Ringer, 1995b. *Acute and Subchronic Toxicity of Naturally Weathered Exxon Valdez Crude Oil in Mallards and Ferrets;* Environmental Toxicology and Chemistry, 14:1941-1950.

Snoeyink, V.L., and D. Jenkins, 1980. *Water Chemistry*; John Wiley and Sons, Inc., New York.

Spacie, A., and J.L. Hamelink, 1985. *Bioaccumulation in Fundamentals of Aquatic Toxicology;* G.M Rand and S.R. Petrocelli, eds., McGraw-Hill, New York, pp. 495-525.

Spann, J.W., R.G. Heath, J.F. Kreitzer, and L.N. Locke, 1972. *Ethyl Mercury p-Toluene Sulfonanilide: Lethal and Reproductive Effects on Pheasants;* Science, 175:328-331. In: EPA, 1995. *Great Lakes Water Quality Initiative Criteria Documents for the Protection of Wildlife: DDT, Mercury, 2,3,7,8 TCDD, and PCBs,* (EPA-820-B-95-008), March 1995.

Stevens, J.B., 1992. *Disposition of Toxic Metals in the Agricultural Food Chain; Steady-State Bovine Tissue Transfer Factors;* Environmental Science and Technology, 25(7): 1289-1294.

Stubblefield, W.A., G.A. Hancock, H.H. Prince, and R.K. Ringer, 1995a. *Effects of Naturally Weathered Exxon Valdez Crude Oil on Mallard Reproduction*; Environmental Toxicology and Chemistry, 14:1951-1960.

Stubblefield, W.A., G.A. Hancock, W.H. Ford, and R.K. Ringer, 1995b. *Acute and Subchronic Toxicity of Naturally Weathered Exxon Valdez Crude Oil in Mallards and Ferrets*; Environmental Toxicology and Chemistry, 14:1941-1950.

Sullivan, K.M., 1991. *Corrective Action for Containing and Controlling Groundwater Contamination;* Lecture Notes, National Water Well Association Short Course.

Sundstrom, C., W.G. Hepworth, and K.L. Diem, 1973. *Abundance, Distribution, and Food Habits of the Pronghorn;* Wyoming Game and Fish Commission, Bulletin No. 12.

Suter, G.W., 1990. *Endpoints for Regional Ecological Risk Assessment;* Environmental Management, 14: 9-23.

Suter, G.W, 1993. *Ecological Risk Assessment*; Lewis Publishers, Chelsea, MI.

Sutton and Nelson, 1937. *Proceedings of the Society for Experimental Biology and Medicine;* 36:211-214.

TEAD Environmental Management Division, 1993. *TEAD South Area: Storm Water Pollution Prevention Plan (revision 1)*; Directorate for Industrial Risk Management, Environmental Management Division, TEAD, Utah.

Thaxton, P., C.R. Parkhurst, and L.A. Cogburn, 1973. *Reproductive Dysfunction and Abnormal Mating Behavior of Japanese Quail as Caused by Mercury*; In: *Heavy Metals in the Environment;* Environmental Health Perspectives, June 1973.

Thaxton, P., C.R. Parkhurst, L.A. Cogburn, and P.S. Young, 1975. *Adrenal Function in Chickens Experiencing Mercury Toxicity;* Poultry Science, 54:578-584.

Thomann, R.V., 1981. *Equilibrium Model of Fate of Microcontaminants in Diverse Aquatic Food Chains;* Canadian Journal of Fisheries and Aquatic Science; 38(280-296).

Travis, C.C., and A.D. Arms, 1988. *Bioconcentration of Organics in Beef, Milk, and Vegetation;* Environmental Science and Technology, 22:271-274.

USATHAMA (U. S. Army Toxic and Hazardous Materials Agency), 1979. *Installation Assessment of Tooele Army Depot;* Report No. 141, U.S. Army Toxic and Hazardous Materials Agency, Aberdeen Proving Ground, Maryland.

USATHAMA, 1987. *Geotechnical Requirements for Drilling, Monitor Wells, Data Acquisition, and Reports;* U.S. Army Toxic and Hazardous Materials Agency, Aberdeen Proving Ground, Maryland.

USATHAMA, 1990. *Quality Assurance Program;* PAM 11-41, Rev. 0, U.S. Army Toxic and Hazardous Materials Agency, Aberdeen Proving Ground, Maryland.

USEPA (U.S. Environmental Protection Agency), 1980. *Interim Guidelines and Specifications for Preparing Quality Assurance Project Plans;* QAMS-005/80, U.S. Environmental Protection Agency, Office of Monitoring Systems and Quality Assurance.

USEPA, 1985. *Compilation of Air Pollutant Emission Factors (AP-42), 4th Edition;* U.S. Environmental Protection Agency, Office of Air Quality, Planning, and Standards, Research Triangle Park, North Carolina.

USEPA, 1987. *Data Quality Objectives for Remedial Response Activities, Development Process;* EPA/540/6-87/003, U.S. Environmental Protection Agency, Washington, D.C.

USEPA, 1989a. *Statistical Analysis of Groundwater Monitoring Data at RCRA Facilities, Interim Final Guidance;* EPA/530-SW-89-026, U.S. Environmental Protection Agency, Office of Solid Waste and Emergency Response, Washington, D.C.

USEPA, 1989b. *Risk Assessment Guidance for Superfund: Volume I, Human Health Evaluation Manual*; EPA/540/1-89/002, Office of Emergency and Remedial Response, Washington, D.C.

USEPA, 1989c. *Risk Assessment Guidance for Superfund: Volume II, Environmental Evaluation Manual, Interim Final*; EPA/540/1-89/001, U.S. Environmental Protection Agency, Office of Emergency and Remedial Response, Washington, D.C.

USEPA, 1989d. *Exposure Factors Handbook;* EPA/600/8-89/043, U.S. Environmental Protection Agency, Office of Health and Environmental Assessment, Washington, D.C.

USEPA, 1991. *Risk Assessment Guidance for Superfund, Volume I: Human Health Evaluation Manual, Supplemental Guidance-Standard Default Exposure Factors, Interim Final;* OSWER Directive 9285.6-03, U.S. Environmental Protection Agency, Office of Emergency and Remedial Response, Washington, D.C.

USEPA, 1992a. *Dermal Exposure Assessment, Principals and Applications, Interim Final;* EPA/600/8-91/011B, U.S. Environmental Protection Agency, Washington, D.C.

USEPA, 1992b. *Health Advisory for Isopropyl Methyl Phosphonic Acid;* U.S. Environmental Protection Agency, Office of Water, Washington, D.C.

USEPA, 1993. *Wildlife Exposure Factors Handbook (Volumes 1 and 2)*; Office of Research and Development, USEPA, Washington, D.C. EPA/600-R-93/187a and b.

USEPA, 1995. *Health Effects Assessment Summary Tables (HEAST);* FY-1995 Annual and FY-1995 Supplement. U.S. Environmental Protection Agency, Office of Emergency and Remedial Response, Washington, D.C.

USEPA, 1997. *Integrated Risk Information System (IRIS)* (Data base); U.S. Environmental Protection Agency, Office of Research and Development.

Venugopal, B. and T.D. Luckey, 1978. *Metal Toxicity in Mammals: Chemical Toxicity of Metals and Metalloids*; Volume II. Plenum Press, NY.

Weber, W., 1987. *Colorado Flora: Western Slope;* Colorado Associated University Press, Boulder, Colorado.

Welsh, S., and others, 1987. *Utah Flora;* Brigham Young University Press, Provo, Utah.

Weston, 1991. *Tooele Army Depot-South Area Remedial Investigation, Final Remedial Investigation Report, Volume I;* Prepared for U.S. Army Toxic and Hazardous Materials Agency, Aberdeen Proving Ground, (Edgewood Area), Maryland.

Wiseman, J., 1987. *Feeding of Nonruminant Livestock*; Butterworths, Boston, MA. pp. 41-201.

Whitworth, M.R., G.W. Pendleton, D.J. Hoffman, and M.B. Camardese, 1991. *Effects of Dietary Boron and Arsenic on the Behavior of Mallard Ducklings*; Environmental Toxicology and Chemistry, 10:911-916.

WHO, 1990a. *Beryllium*; Environmental Health Criteria 106, Geneva, Switzerland

WHO, 1992a. *Di-ethylhexyl phthalate*; Environmental Health Criteria 131, Geneva, Switzerland.

Comments on the TAD South Area Final Phase II RFI SWMUs 13 and 17 (August, 1995), Human Health and Ecological Risk Assessment
Chris Bittner, Utah DSHW

General Comments

1. In general, the human health risk assessment was conducted and presented in accordance with current guidance. As noted in the specific comments, some exposure parameters are not consistent with a reasonable maximum exposure (RME) and the risk calculations should be revised using RME parameters. In anticipation that some of the calculations will change, the actual calculations in the appendices were not reviewed for this draft.

2. The ecological risk assessment has some deficiencies. Comments were provided in the letter dated _____ and are not repeated here. Please revise the ecological assessment in accordance the issues discussed at the _____ TRC meeting.

Specific Comments

1. In the tables that illustrate exposure point concentrations, the footnotes note that ND indicates not detected. Were the metals not detected or not detected above background?

2. A child resident scenario is evaluated because children may engage in activities that result in a higher exposure than an adult. To use subchronic toxicity values to evaluate the child may circumvent the intent of evaluating this sensitive population. At minimum, please demonstrate that using subchronic toxicity values is at least as protective as evaluating a seven year, chronic exposure (child for six years, adult for one).

3. The text indicates that when USEPA Health Criteria are not currently available, it was not feasible to evaluate human health impacts. Why weren't these compounds qualitatively evaluated? For instance, the impact of a lack of a toxicity value for methyl-n-butyl ketone is different than the lack of a toxicity value for bicarbonate. The text should note, where appropriate, when the compound is of low toxicity and is unlikely to be of concern. At minimum, a SWMU specific discussion of uncertainties in the risk assessment should include this.

4. p. 6-4, 6.1.2.1.2. The text indicates that a construction worker was included when chemical were measured below 1 foot bgs. Why isn't the absence of data for below 1 foot [illegible] data gap?

5. p. 6-5. Engineering controls. A baseline risk assessment should be conducted assuming absence of engineering controls. If a site with engineering controls is recommended for no further action, what mechanism ensures that these controls remain in place?

6. p. 6-31. Groundwater. Please define the terms upper-bound and high-end. Weren't values based on the upper confidence level of the MEAN?

7. p. 6-32. Vegetable Consumption. A modifying factor of 0.10 to estimate uptake into fruits and tubers is not supported by the current empirical evidence and theory of plant uptake of organic chemicals (e.g., Bell, 1992). For some chemicals, an uptake factor for leafy plants (Equation 6-2) will likely underestimate chemical uptake into the root portion of plants. In the absence of additional information, a modifying factor is not justified.

8. Table 6-3 and 6-4. Summary of Exposure Factors.

a) On-site Worker Exposure Frequency. Is the assumption that a worker spends time at different SWMUs based on site-specific information or a hypothetical worst case? The worker scenario should reflect site-specific conditions and be protective for the majority of workers now and in the future (UAC R315-101-5(5.2)(b)(2). Is assuming that a worker splits time between sites protective of the majority of workers? Please provide a discussion that demonstrates that this scenario is protective for evaluating carcinogenic and noncarcinogenic exposures.

b) Is the soil ingestion rate for workers an RME parameter? The default soil ingestion is 50 mg/day (do the workers, such as maintenance personnel, engage in soil-intrusive activities resulting in a higher soil ingestion rate?) which is based on an 8 hour day. This results in an ingestion rate of 250 mg/week. To adjust for a 10 hour day, the soil ingestion should be 62.5 mg/day.

c) Depot worker inhalation rate. The rate of 0.8 m^3/hour is based on the EPA (1991) default of 20 m^3/day. This is an incorrect application because it assumes that the rate is for a 24 hour day. To convert to an hourly rate, divide by an 8-hour day (2.5 m^3/hour).

d) Construction Worker Skin Surface Area. The value of 2,000 cm^2 assumes that the worker is exposed only on the head and hands. Given that the construction activity is assumed to be one month a year, and that this month is likely in the summer (in the desert), the value of 2,000 cm^2 appears to underestimate the exposure. Assuming the construction worker is clothed in shoes, long pants, and short sleeve shirt results in an estimate of 4,590 cm^2 (On-Site Worker, Table 6-3).

e) Absorption. The absorption values for soil to skin were assumed to be 0.1% for inorganics and 1% for organics. While no specific values are recommended by EPA, the values of 0.1% and 1% are at the lower end of the reported values. Based on the data presented in EPA (1992), a value of 10% is reasonably conservative for organics. On page 6-40, it indicates that these are EPA Region VIII recommended values. Please provide either documentation or the contact person to verify these values.

f) What is the rationale for the skin surface area values for residents? The adult value is a central tendency and not an RME (5,800 cm^2) for soil or bathing (23,000 cm^2) (Table 8-6, EPA, 1992). How was the child skin-surface area for bathing derived?

The 95th percentile for male children, ages three to six years, is 8,800 cm^2 (Table 8-4, EPA 1992).

9. p. 6-74, Section 6.1.5.4; (also 6-39, 6-40, 6-76). The text is erroneous in interpreting the value used for the exposure point concentration. The 95% upper confidence limit of the mean was used to estimate the exposure point concentration (concentration term). The concentration term is intended to be an average value, and not a high end, upper-bound, or percentile value (it is correct to say that it is high-end estimate of the mean). The statistical definition is that with 95% confidence (5% chance of an error), the true mean soil concentration is between the lower and upper confidence limit. The upper confidence limit is selected as the most conservative estimate of the mean. No conclusion can be made regarding whether the true mean is likely closer to the upper or lower confidence limit. In the instances where the maximum detected concentration was less than the 95% UCL, the exposure point concentration may be an underestimate of the mean. Please clarify the text. Additional information can be found in EPA, 1992a

10. p. 6-41, Worker Inhalation of Particulates. EPA guidance does not recommend the that a deposition fraction term be used for default scenarios. RfC's and inhalation slope factors are based on an administered concentration. Unless site-specific information is available that indicates that the particulates at SWMUs 13 and 17 are less bioavailable than the chemical used in the toxicological experiments for the IRIS values, no deposition fraction should be used.

11. p. 6-42, On-Site Construction Worker. What is the impact of assuming that the 30-day exposure frequency is not consecutive days?

12. p. 6-46. Permeability Coefficient. Please use the permeability coefficients recommended in EPA (1992). The default for inorganics, the same as for water, is 10^{-3} cm^2/hour (pages 5-38 and 5-97). For organics, use the values in Table 5-7, or one of the other methods in EPA (1992).

13. p. 6-46. Administered vs. Absorbed Dose for Dermal Exposures. Adjusting the RfD to calculate an absorbed dose is consistent with RAGS (EPA, 1989). In Appendix A of RAGS, a default absorption value of 5 percent is recommended, not the values cited on p. 6-46 of 100 percent for VOCs and 50 percent for SVOCs and inorganics. However, information collated since RAGS was issued suggests that using these values will not result in a significant underestimation of risk from dermal exposures. Why wasn't this procedure used for dermal exposures to soil?

14. Table 6-5 through 6-10.

a) The reference indicates that the toxicity values were updated from IRIS in 1994. Please check the values to ensure that they are up-to-date. The ECAO has an interim oral RfD of 6×10^{-3} mg/kg-day for trichloroethene, and IRIS has an oral slope factor of 8.4×10^{-2} $(mg/kg\text{-}day)^{-1}$ of dibromochloromethane. Please quantify the risks associated with these compounds.

b) Using sodium fluoracetate as a surrogate for fluoracetate is acceptable. However, the toxicity value for fluoracetate should be adjusted for the difference in molecular weight.

c) RAGS (page 7-15) and UAC R315-101-5(5.3)(a)(6) discusses the hierarchy of sources of toxicity information. If a toxicity value is unavailable from IRIS, HEAST, or ECAO alternative values may be derived using the EPA methodology. If no value is available, the data gap must be evaluated in the discussion of uncertainties. In the uncertainties, a comparison to regulatory standards (which may be based on management or technological considerations) may be helpful. It is inappropriate to substitute other values in the quantitative calculations of the excess lifetime cancer risk or noncancer health effects (e.g. as was done for copper and lead).

15. p. 6-73, Section 6.1.5.1. TICs should not be "disregarded". Please provide a discussion of TICs that includes the identification and concentration, the potential implications to human health and the environment, why the TIC should or should not be considered a contaminant of concern, and whether additional sampling is warranted (see RAGS, Section 5.6).

16. p. 6-70, Section 6.1.5.2. Please expand the discussion regarding the negligible exposures via the ingestion of contaminated game. For instance, the exposures are being calculated for ingestion of contaminated beef which should be a conservative estimate for exposure by ingestion of contaminated game.

17. p. 7-90, Table 7-23. For the current land use scenarios, it was assumed that asphalt would prevent current exposures (samples 13 3X-04-1, 05-1, 06-1, 07-1). What is the impact to the risk estimates for the current land use scenarios.

18. p. 7-186, Section 7.9.1. What is the source of the elevated alkalinity/bicarbonate in 13SH-01 through 13SH-04?

19. p. 7-201, Section 7.10.3.1. Why was a construction worker deemed unlikely for this site? A construction worker likely built the lagoons. What if the lagoons needed to be enlarged or repaired? Please evaluate the construction worker for the waste-water lagoons.

20. Section 7.10. How can the gross alpha and beta be interpreted in the absence of background values or without identifying the element responsible? It is acceptable to assume they are present above background. If the gross alpha and beta are assumed to be present above background, they must be evaluated. Typically, gross alpha and beta are measured as a screening analysis. Is additional sampling warranted?

21. p. 7-232. Ground Water Analytical Data. The discrepancies between the 1991 and 1993 data set are disturbing. While the 1993 data set may be the most defensible, it is the least protective of human health. Additional sampling rounds are needed to provide any confidence that the nature and extent of contamination has been defined. The additional sampling rounds should include as analytes all substances that were detected in previous

rounds (e.g., 1,2-dimethyl benzene, di-, tri, and methylnaphthalene). Given the high acute toxicity of fluoracetic acid, at minimum, samples should be analyzed for formic acid. Otherwise, this is just an unconfirmed hypothesis that the fluoracetic acid detected in 1991 was really formic acid.

22. p. 7-273. The data and rationale presented on p. 7-266 do not support that arsenic is present at background concentrations. The text on p. 7-273 indicates that arsenic is likely present at background concentrations. Please reconcile.

23. p. 7-273. How can "_arsenic and thallium combined account for approximately 98 percent of the total groundwater cancer risk_" when thallium is a Class D carcinogen?

24. p. 8-29. Section 8.4.2 Nature and Extent of Contamination. In surface soil, arsenic was detected above background in both samples, chromium in 2 out 5 samples, copper in 5 out 5, lead in 4 out of 5, and zinc in the single sample (Table 8-14). Yet the text indicates that these are likely present at background? What is the basis for the supposition that this is "clean fill"? Why is the placement of fill not considered a part of site operations?

REFERENCES

Bell, R.M. 1992. Higher Plant Accumulation of Organic Pollutants from Soils. EPA/600/R-92/138. June, 1992

EPA, 1991. Human Health Evaluation Manual, Supplemental Guidance: "Standard Default Exposure Factors" OSWER Directive 9258.6-03. March 25, 1991

EPA, 1992. Dermal Exposure Assessment: Principles and Applications. Interim Report. EPA/600/8-91/011B. January, 1992

EPA, 1992a. Supplemental Guidance to RAGS: Calculating the Concentration Term. OSWER Publication 9285.7-088. May, 1992.

Comment	Response to Comment
State of Utah	
General Comments	
1. In general, the human health risk assessment was conducted and presented in accordance with current guidance. As noted in the specific comments, some exposure parameters are not consistent with a reasonable maximum exposure (RME) and the risk calculations should be revised using RME parameters. In anticipation that some of the calculations will change, the actual calculations in the appendices were not reviewed for this draft.	Comments are noted and the appropriate changes have been made to the document. In addition, a 100-percent QC of all calculations was conducted for this revision.
2. The ecological risk assessment has some deficiencies. Comments were provided in the letter dated October 10, 1996, and are not repeated here. Please revise the ecological assessment in accordance with the issues discussed at the October 31, 1996, TRC meeting.	As noted on pages 8 through 10 of this response document, SWMU 13 and 17 sites have been re-evaluated for ecological risk using updated toxicological information. Further, several sites at SWMU 13 are totally enclosed within security fencing, are void of vegetation, and are continually frequented by humans. As a result, the ecological risk at these sites is considered minimal (Fuel Spill Site, Underground Storage Tank Site, 3X Yard, Pavement Perimeter Site, and Sodium Hydroxide Spill Site). Current plans are for these sites to remain as Army facilities and therefore, no further investigations appear to warranted.
Specific Comments	
1. In the tables that illustrate exposure point concentrations, the footnotes note that "ND" indicates not detected. Were the metals not detected or not detected above background?	To clarify this issue, the tables have been changed such that <Bkgd indicates detected but less than background and ND indicates not detected.
2. A child resident scenario is evaluated because children may engage in activities that result in a higher exposure than an adult. To use subchronic toxicity values to evaluate the child may circumvent the intent of evaluating this sensitive population. At minimum, please demonstrate that using subchronic toxicity values is at least as protective as evaluating a seven year, chronic exposure (child for six years, adult for one).	The health criteria (e.g., chronic/subchronic RfDs) are developed to be protective of the most sensitive subgroups in the population. Subchronic criteria are typically used for shorter duration if additional health effects could occur and/or if chemical bioaccumulation is a factor in expression of the toxic endpoint.
3. The text indicates that when USEPA Health Criteria are not currently available, it was not feasible to evaluate human health impacts. Why weren't these compounds qualitatively evaluated? For instance, the impact of a lack of toxicity value for methyl-n-butyl ketone is different than the lack of a toxicity value for bicarbonate. The text should note, where appropriate, when the compound is of low toxicity and is unlikely to be of concern. At minimum, a SWMU-specific discussion of uncertainties in the risk assessment should include this.	Qualitative discussions of uncertainties were added to the text for each site.

Comment	Response to Comment
4. p. 6-4, 6.1.2.1.2. The text indicates that a construction worker was included when chemicals were measured below one foot bgs. Why isn't the absence of data for below one foot bgs a data gap?	With the exception of the Sewage Lagoons, all sites had both surface and subsurface samples collected and as a result, no data gap exists. The sentence was revised to state that ". . . where contamination was identified in subsurface samples (below 1 foot bgs)."
5. p. 6-5 Engineering controls. A baseline risk assessment should be conducted assuming an absence of engineering controls. If a site with engineering controls is recommended for no further action, what mechanism ensures that these controls remain in place?	The reviewer is correct. The text has been revised to remove the references to engineering controls or access restrictions. The individual conceptual site models, however, are correct and the baseline risk assessment was conducted for all complete pathways.
6. p. 6-31. Groundwater. Please define the terms "upper-bound" and "high-end." Weren't values based on the upper confidence level of the MEAN?	Text was added/modified to clarify that the exposure point concentrations represent either the 95 percent UCL of the mean or the maximum detected concentration, whichever was lower.
7. p. 6-32. Vegetable Consumption. A modifying factor of 0.10 to estimate uptake into fruits and tubers is not supported by the current empirical evidence and theory of plant uptake of organic chemicals (e.g., Bell, 1992). For some chemicals, an uptake factor for leafy plants (Equation 6-2) will likely underestimate chemical uptake into the root portion of plants. In the absence of additional information, a modifying factor is not justified.	Current literature was reviewed to determine the appropriate plant uptake factors for organics and the uptake factors were revised accordingly.

Comment	Response to Comment
8. Table 6-3 and 6-4. Summary of Exposure Factors. a) On-site Worker Exposure Frequency. Is the assumption that a worker spends time at different SWMUs based on site-specific information or a hypothetical worst case? The worker scenario should reflect site-specific conditions and be protective for the majority of workers now and in the future (UAC R315-101-5(5.2)(b)(2). Is assuming that a worker splits time between sites protective of the majority of workers? Please provide a discussion that demonstrates that this scenario is protective for evaluating carcinogenic and noncarcinogenic exposures.	Since each of the sites within a given SWMU are in close proximity to each other, it is our view that a worker at each SWMU would likely "visit" each site each day. Mathematically, this was handled by dividing the work year by the number of sites at each SWMU. Text has been added to clarify this. In reality, the majority of the workers spend their time inside the facility buildings and are not exposed to any of the contaminated sites during a normal work day. Assuming exposure to each site adds to the conservative nature of the risk assessment.
b) Is the soil ingestion rate for workers an RME parameter? The default soil ingestion is 50 mg/day (do the workers, such as maintenance personnel, engage in soil-intrusive activities resulting in a higher soil ingestion rate?) which is based on an 8 hour day. This results in an ingestion rate of 250 mg/week. To adjust for a 10 hour day, the soil ingestion should be 62.5 mg/day.	EPA guidance states that the 50 mg/day value is to be used in conjunction with an exposure frequency of 250 days/year (5 days per week for 50 weeks per year), or a total yearly soil ingestion rate of 12,500 mg. In order to be consistent with this guidance, since the DCD workers are assumed to work 208 days per year (4 days per week for 52 weeks per year), their daily soil ingestion rate should have a value of approximately 60 mg/day. This change has been made.
c) Depot worker inhalation rate. The rate of 0.8 m^3/hour is based on the EPA (1991) default of 20 m^3/day. This is an incorrect application because it assumes that the rate is for a 24 hour day. To convert to an hourly rate, divide by an 8-hour day (2.5 m^3/hour).	Comment noted. Correction was made to the document.
d) Construction Worker Skin Surface Area. The value of 2,000 cm^2 assumes that the worker is exposed only on the head and hands. Given that the construction activity is assumed to be one month a year, and that this month is likely in the summer (in the desert), the value of 2,000 cm^2 appears to underestimate the exposure. Assuming the construction worker is clothed in shoes, long pants, and short sleeve shirt results in an estimate of 4,590 cm^2 (On-Site Worker, Table 6-3).	Comment noted. Correction was made to the document.

Comment	Response to Comment
e) Absorption. The absorption values for soil to skin were assumed to be 0.1% for inorganics and 1% for organics. While no specific values are recommended by EPA, the values of 0.1% and 1% are at the lower end of the reported values. Based on the data presented in EPA (1992), a value of 10% is reasonably conservative for organics. On page 6-40, it indicates that these are EPA Region VIII recommended values. Please provide either documentation or the contact person to verify these values.	The 1.0% and 0.1% skin absorption values for organics and inorganics, respectively, were recommended for use by USEPA Region IV (New Interim Region IV Guidance, February 1992) for determining risks associated with dermal exposure to contaminated soils. These values take into account the soil matrix effect.
f) What is the rationale for the skin surface area values for residents? The adult value is a central tendency and not an RME (5,800 cm^2) for soil or bathing (23,000 cm^2) (Table 8-6, EPA, 1992). How was the child skin-surface area for bathing derived? The 95th percentile for male children, ages three to six years, is 8,800 cm^2 (Table 8-4, EPA 1992).	Skin surface area is dependent upon body weight. Since central tendency body weights were used for both adult and child receptors, central tendency surface areas are also. Text was added to clarify this point.
9. p. 6-74, Section 6.1.5.4; (also 6-39, 6-40, 6-76). The text is erroneous in interpreting the value used for the exposure point concentration. The 95% upper confidence limit of the mean was used to estimate the exposure point concentration (concentration term). The concentration term is intended to be an average value, and not a high end, upper-bound, or percentile value (it is correct to say that it is high-end estimate of the mean). The statistical definition is that with 95% confidence (5% chance of an error), the true mean soil concentration is between the lower and upper confidence limit. The upper confidence limit is selected as the most conservative estimate of the mean. No conclusion can be made regarding whether the true mean is likely closer to the upper or lower confidence limit. In the instances where the maximum detected concentration was less than the 95% UCL, the exposure point concentration may be an underestimate of the mean. Please clarify the text. Additional information can be found in EPA, 1992a.	Text has been clarified.
10. p. 6-41, Worker Inhalation of Particulates. EPA guidance does not recommend that a deposition fraction term be used for default scenarios. RfC's and inhalation slope factors are based on an administered concentration. Unless site-specific information is available that indicates that the particulates at SWMUs 13 and 17 are less bioavailable than the chemical used in the toxicological experiments for the IRIS values, no deposition fraction should be used.	Text was added to clarify this issue. If a criterion is based on particulate inhalation experiments, then this term was set at 1.0 for that chemical.
11. p. 6-42, On-Site Construction Worker. What is the impact of assuming that the 30-day exposure frequency is not consecutive days?	If 30 consecutive days are assumed, then the averaging time becomes 30 days rather than 365 days annually. This change would lead to non-cancer exposure doses that are approximately 12 times higher than what are currently calculated.

Comment	Response to Comment
12. p. 6-46. Permeability Coefficient. Please use the permeability coefficients recommended in EPA (1992). The default for inorganics, the same as for water, is 10^{-3} cm^2/hour (pages 5-38 and 5-97). For organics, use the values in Table 5-7, or one of the other methods in EPA (1992).	The default PC for inorganic chemicals was changed to 0.001 cm^2/hour. The equation for calculating the dermal PC for organics (Equation 6-17) was taken from the scientific literature and is based on a regression analysis of experimentally measured chemical uptake through skin, i.e., empirical data. The comparable equation provided in USEPA's *Dermal Exposure Guidance: Principles and Applications* was not used since it is theoretical in nature. Regarding the approach provided in the EPA document, the EPA itself states (on p. 5-52), "...This seems counterintuitive and raises concerns that the model may be overly conservative. Lack of data makes validation of the model very difficult..." It is our position that the equation in the referenced literature is more scientifically defensible than the equation presented in the EPA document.
13. p. 6-46. Administered vs. Absorbed Dose for Dermal Exposures. Adjusting the RfD to calculate an absorbed dose is consistent with RAGS (EPA, 1989). In Appendix A of RAGS, a default absorption value of 5 percent is recommended, not the values cited on p. 6-46 of 100 percent for VOCs and 50 percent for SVOCs and inorganics. However, information collated since RAGS was issued suggests that using these values will not result in a significant underestimation of risk from dermal exposures. Why wasn't this procedure used for dermal exposures to soil?	Normalizing dermal exposure doses to the oral route was not conducted for soil contact since Region IV toxicologists told us that their matrix effect approach (which we used in the risk assessment) already corrects for this route-to-route extrapolation.

Comment	Response to Comment
14. Table 6-5 through 6-10. a) The reference indicates that the toxicity values were updated from IRIS in 1994. Please check the values to ensure that they are up-to-date. The ECAO has an interim oral RfD of 6 x 10^{-3} mg/kg-day for trichloroethene, and IRIS has an oral slope factor of 8.4 x 10^{-2} (mg/kg-day)$^{-1}$ of dibromochloromethane. Please quantify the risks associated with these compounds.	All health criteria were updated for this version of the document. The risks/hazards for both of the listed chemicals were quantitatively evaluated.
b) Using the sodium fluoracetate as a surrogate for fluoracetate is acceptable. However, the toxicity value for fluoracetate should be adjusted for the difference in molecular weight.	Comment noted. Text and criteria have been modified.
c) RAGS (page 7-15) and UAC R315-101-5(5.3)(a)(6) discusses the hierarchy of sources of toxicity information. If a toxicity value is unavailable from IRIS, HEAST, or ECAO alternative values may be derived using the EPA methodology. If no value is available, the data gap must be evaluated in the discussion of uncertainties. In the uncertainties, a comparison to regulatory standards (which may be based on management or technological considerations) may be helpful. It is inappropriate to substitute other values in the quantitative calculations of the excess lifetime cancer risk or noncancer health effects (e.g., as was done for copper and lead).	These values are <u>not</u> substitutes for existing criteria/standards. Rather, they are exposure doses derived from these criteria/standards using default exposure parameters. Region III and other regions have used this approach in driving their screening values; e.g., RBCs.
15. p. 6-73, Section 6.1.5.1. TICS should not be "disregarded." Please provide a discussion of TICs that includes the identification and concentration, the potential implications to human heath and the environment, why the TIC should or should not be considered a contaminant of concern, and whether additional sampling is warranted (see RAGS, Section 5.6).	Text was included to be consistent with RAGs.
16. p. 6-70, Section 6.1.5.2. Please expand the discussion regarding the negligible exposures via the ingestion of contaminated game. For instance, the exposures are being calculated for ingestion of contaminated beef which should be a conservative estimate for exposure by ingestion of contaminated game.	Additional text has been added to the document.
17. p. 7-90, Table 7-23. For the current land use scenarios, it was assumed that asphalt would prevent current exposures (samples 13 3X-04-1, 05-1, 06-1, 07-1). What is the impact to the risk estimates for the current land use scenarios?	Since the asphalt paving is in place, current worker exposure to these sample locations cannot occur. Therefore, the effect of including these data in the calculations is moot. Please note, however, that all of the data were used in the future worker scenario; i.e., it was assumed the asphalt was no longer present (see footnote in Table 7-23).
18. p. 7-186, Section 7.9.1. What is the source of the elevated alkalinity/bicarbonate in 13SH-01 through 13SH-04?	The likely source is an intermediate byproduct of the chemical reaction between the sodium hydroxide and natural carbonates in the soil.

Comment	Response to Comment
19. p. 7-201, Section 7.10.3.1. Why was a construction worker deemed unlikely for this site? A construction worker likely built the lagoons. What if the lagoons needed to be enlarged or repaired? Please evaluate the construction worker for the waste-water lagoons.	To address this concern, risk estimates were added for a future construction worker at the wastewater lagoons.
20. Section 7.10. How can the gross alpha and beta be interpreted in the absence of background values or without identifying the element responsible? It is acceptable to assume they are present above background. If the gross alpha and beta are assumed to be present above background, they must be evaluated. Typically, gross alpha and beta are measured as a screening analysis. Is additional sampling warranted?	Based on process knowledge, no operations at CAMDS involved the use, storage, or disposal of radionuclides. Uranium and its daughter products (i.e., radium-226 and 228) are the most likely sources of the gross alpha and beta activity. A review of facility-wide groundwater data shows that radionuclides are widespread and highly variable throughout the entire basin. No attempt has been made to date to establish background values since all investigations have concluded that the radionuclides are naturally occurring. The reviewer is correct concerning gross alpha and beta measurements being primarily used as a screening analysis. The uranium data, however, were used to assess potential risks for the Sewage Leach Ponds Site. No additional sampling is warranted.
21. p. 7-232. Ground Water Analytical Data. The discrepancies between the 1991 and 1993 data set are disturbing. While the 1993 data set may be the most defensible, it is the least protective of human health. Additional sampling rounds are needed to provide any confidence that the nature and extent of contamination has been defined. The additional sampling rounds should include as analytes all substances that were detected in previous rounds (e.g., 1,2-dimethyl benzene, di-, tri-, and methylnaphthalene). Given the high acute toxicity of fluoracetic acid, at minimum, samples should be analyzed for formic acid. Otherwise, this is just an unconfirmed hypothesis that the fluoracetic acid detected in 1991 was really formic acid.	The Corps of Engineers is continuing to perform semi-annual sampling of SWMU 13 wells. As reported in the January 30, 1997 TRC meeting, no agent breakdown products have been detected, thereby supporting the 1993 data. The April 1996 sampling showed that arsenic, thallium, benzene, xylene, and TPH are the contaminants present. Sampling will be performed again in April 1997. Results will be evaluated further as part of the CMS conducted by Dames & Moore.
22. p. 7-273. The data and rationale presented on p. 7-266 do not support that arsenic is present at background concentrations. The text on p. 7-273 indicates that arsenic is likely present at background concentrations. Please reconcile.	Sentence on page 7-273 referring to arsenic as background was deleted.
23. p. 7-273. How can arsenic and thallium combined account for approximately 98 percent of the total groundwater cancer risk when thallium is a Class D carcinogen?	Comment noted. Text was revised.
24. p. 8-29. Section 8.4.2 Nature and Extent of Contamination. In surface soil, arsenic was detected above background in both samples, chromium in two out of five samples, copper in five out of five, lead in four out of five, and zinc in the single sample (Table 8-14). Yet the text indicates that these are likely present at background? What is the basis for the supposition that this is "clean fill?" Why is the placement of fill not considered a part of site operations?	References to the metals being naturally occurring were removed. The word "clean" was removed when referencing the fill. The important fact remains that the referenced metals were carried forward and evaluated in the risk assessment regardless of origin.

Comment	Response to Comment
Ecological Risk Assessment Comments from Previous State of Utah Comment Package Dated October 10, 1996.	
2. The ecological risk assessment has some deficiencies in the methodology when compared to the Framework for Ecological Risk Assessment (EPA 1992). The problem formulation is complete with the exception of endpoint selection. The connection between the measurement and assessment endpoints is tenuous. The measurement endpoints should be stated in the form of a testable hypothesis. If the endpoints are to be weighted differently, the weighting and rationale should be presented in the methodology. The result of this deficiency is that the conclusions (interpretation of ecological significance) are not supported by the analysis.	Table 6-13 "Ecological Endpoints" (previously Table 6-12) was revised as a significant aspect of the revised ecological risk assessment. The measurement endpoint is expressed in terms of assessment endpoint parameters, i.e., concentrations of COPCs in surface soil and surface water. A testable hypothesis is proposed as an integral part of the endpoint decision parameter. Conclusions in the SWMU/site sections relate directly to the decision parameter.
3. Lethality was selected as the toxicological endpoint for ecological receptors. The toxicological benchmarks, which need documentation, are based on median lethal doses or concentrations for acute exposures. This results in the conclusion of no-adverse-effects if up to 50% of the population is predicted to be killed by exposure to COPCs. This is not a protective approach. Typically, lowest-adverse-effects levels or no-observed-adverse-effects levels (LOAELs) and NOAELs) are selected as toxicological benchmarks. For the State to accept the use of median lethal doses, the report must demonstrate how these are protective.	Protective screening level hazard quotients used in this Revised Final RFI were calculated using EPA Region VIII approved toxicity reference values as established for the TEAD SWERA.
4. Ecological Assessment. Evaluating a limited number of receptors (key species) is acceptable provided that it can be demonstrated that the key species have a higher calculated exposure than the other receptors.	Section 6.2.1.3 (formerly Section 6.2.3.3) was re-written to refine the justification for selection of key receptors. Note that some key receptors were revised because of availability of toxicity and other biological data.
14. Table 6-11. Hazard Rating for Ecological COPCs. The rationale for the non-toxicity criteria in the hazard rating (e.g. flammability and reactivity) is unclear. These appear to be transportation requirements and not related to ecological receptors. What compounds were screened with using the criteria of flammability and reactivity?	This table was revised to be a simple listing of COPCs. The other data was found to be redundant and/or inappropriate. Information concerning biological effects of COPCs is found in Table 6-2. COPC tables under site discussions in Sections 7 and 8 were also revised.
15. Page 6-86, Section 6.2.4. Toxicity Assessment. The rationale for using acute toxicity values is not clear. The ecological endpoints (Table 6-12) include assessment endpoints of significant population reductions in key species and adverse effects to threatened and endangered species. An ecological assessment based on acute LD_{50} values does not provide a measure for these assessment endpoints.	Please see responses to comments 2 and 3 above.

Comment	Response to Comment
16. Section 6.2.5. Please provide the units for the equation variables.	Units are now provided for all equations. Note that the calculation methodolgy has been revised completely for this Revised Final RFI as stated in the following response.
17. Section 6.2.5. Assimilation efficiency and depuration: As noted in the reference for the assimilation efficiency of 0.9 (Spacie et al., 1985; p. 513), this model is highly sensitive to the relative values for assimilation efficiency and depuration. A small error in estimating either coefficient will affect the outcome significantly. If one value is to be used for assimilation efficiency and depuration rate for all COPCs and all receptors, the value must be protective. That is, all COPCs will be absorbed at, or less than, this rate. The ESE (1988) reference for depuration rates is not provided. Because the ESE (1988) document will not be readily available to the DSHW or the public, please include the relevant portions as part of the report. Please expand the discussion in the text to demonstrate the values used are likely conservative.	Calculations of soil ingestion, soil dermal contact, water ingestion and dietary uptake were revised in accordance with the approach used for the TEAD SWERA.
18. The risk characterization and resulting conclusions for ecological receptors are not clear. For instance, Table 8-20 concludes that a decrease in ferruginous hawk population is unlikely. The rationale for this conclusion is not apparent. If the MTL is used to calculate a hazard index, the hazard index for the deer is 50 and the hazard index for the hawk is over 100 at the Drum Storage Site. If LD_{50} values were used to derive the MTLs, then the prediction is that over 50% of the exposed raptor population would be killed and the survivors would likely suffer from non-lethal effects from chronic exposures. The raptor population includes threatened and endangered species, and therefore even individual birds warrant protection. Please revise the conclusions or provide supporting rationale.	As a consequence of modification of endpoints and toxicological values (see responses to comments 2 and 3 above), the results, conclusions, and rationale have been extensively revised Conclusions now consider, among other parameters, accessibility by ecological receptors to SWMU 13 sites as discussed in general comment 2 above (page 1 of this document).
21. Appendix H. Ecological Assessment. Please provide a summary of the exposure parameters, including the source and rationale, used in the calculation of the maximum tolerance levels, of the biomagnification factors, and the toxicity values. For instance, what is the source of the food, soil, and water ingestion rates?	Additional data, including references, have been included in new tables in the methodology section (Section 6.0) of the text. The information previously presented in Appendix H has been removed.
22. Appendix H. Ecological Assessment. Where are footnotes 1 and 2?	This table was removed and data included in the body of the report as part of the overall revision to eco-risk calculations.